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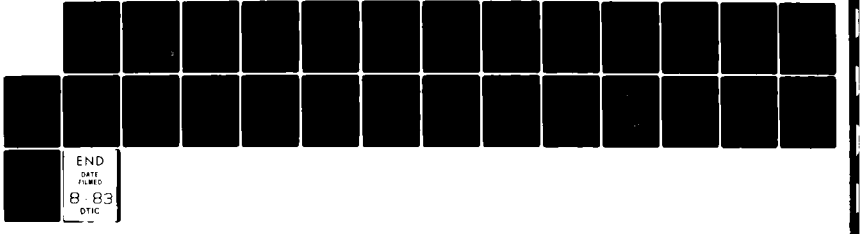

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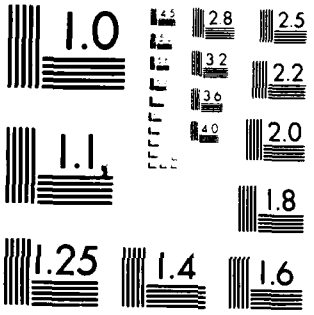

AUDITORY AND NON-AUDITORY EFFECTS OF EXPOSURE TO LOW-FREQUENCY NOISE

FINAL REPORT

JOHN H. MILLS

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reduced to 90 dBA for 8 h. For this condition TTS increased throughout the 8 h exposure. TTS from the 90-dBA exposure for 8 h exceeded the TTS produced by the 24 h exposure at the 84 dBA. Whereas recovery from the 24 h exposure which produced TTS's of 15 dB required as long as 48 h, recovery from the 8 h exposure which produced TTS's of 20 dB required 12-24 h. Thus, the time required for recovery from TTS is determined in part by the duration of exposure. Non-auditory effects included small increases in serum cortisol only during the first hour of exposure. Heart rate, blood pressure, and catecholamines showed small changes which were not statistically significant.

Acoust. Soc. Abstract.

Some Auditory and Non-Auditory Effects of Low-Frequency Noises. John H. Mills, J. David Osguthorpe (Department of Otolaryngology, Medical University of South Carolina, Charleston, SC 29425), C. K. Burdick (U.S. Army Med. Res. and Dev. Command, Fort Detrik, Frederick, MD 21701), J. H. Patterson and B. Mozo (U.S. Army Aeromedical Research Lab, Fort Rucker, AL 36360).

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PART I. AUDITORY EFFECTS

INTRODUCTION AND BACKGROUND

Exposure to noise can injure the ear and produce a hearing loss. The injury and hearing loss can be temporary, permanent, or chronically present for exposures which are repeated. Many military vehicles, particularly those found in the Armor Branch of the Army, produce high-intensity, low-frequency noise, and therefore a risk of hearing loss. Current damage-risk criteria are specified in terms of dBA which de-emphasizes the importance of low-frequency noise. A full understanding of the effects of low-frequency noise on hearing is essential to the development of adequate hearing conservation and material limits for the protection of armored vehicle crews.

With human subjects in laboratory investigations, one is forced (for obvious reasons) to study temporary changes in hearing which are called temporary threshold shifts (TTS). One then attempts to predict permanent effects from temporary effects. Of the facts available about TTS, one new result is that low-frequency noise can produce high-frequency hearing losses (Patterson, et al., 1977; Mills et al., 1979). The frequency region is typically one-half to one octave above the exposure frequency (David et al., 1950). This is a general finding for mammallian ears. A recent series of experiments conducted at the U.S. Army Aeromedical Research Laboratory (USAARL) found an exception to this typical finding (for example, Burdick et al., 1978; Burdick et al., 1977). Chinchillas exposed to high-intensity, low-frequency noise experienced a maximum permanent hearing loss five octaves above the center frequency of the band of noise to which they were exposed. For example, chinchillas exposed to an octave band of noise centered at 63 Hz incurred peak hearing losses at 2000 Hz. In an attempt to determine the generality of this effect to humans, a preliminary study was conducted (Patterson et al., 1977). Human subjects were exposed to octave-band noise centered at 63 Hz at intensity levels of 110 dB SPL (84 dBA) and 120 dB SPL (94 dBA). Exposure durations were restricted to 4 hours and temporary threshold shifts (TTS's) were measured. These results indicated that the low-frequency noise affected the frequency region of 1.0 to 3.0 kHz in humans, results quite similar to those for the chinchilla. Several questions remained unanswered, however. A very important issue concerns the effects produced by longer duration exposures. Others have found that plateaus or asymptotes of threshold shifts in humans typically require noise exposures ranging from 8-12 hours (Mills et al., 1970; Melnick, 1976; and Mills et al., 1979). Consequently, the 4 hour exposure to the low-frequency noise (Patterson et al., 1977) probably produced TTS levels below those that would occur at their asymptotic thrshold shift (ATS) levels. Longer duration exposures are necessary to determine the validity of our initial findings with regard to the frequency showing peak effects and to obtain the ATS levels produced by the 63 Hz octave-band of noise at the same intensity levels used with the chinchillas (Burkick et al., 1978; Burdick et al., 1977).

Whereas the data base for TTS is outstanding for noises with energy concentrated between 500 Hz and 4000 Hz, there are very few data for noises below 500 Hz. The growth and decay of TTS, the relation between TTS at asymptote (ATS) and the level of noise, and the spread of TTS along the frequency dimension need to be established by exposure to octave bands of noise centered at 250, 125 and 63 Hz.

PROCEDURES

Subject

Subjects were 52 male students between the ages of 18-22 who were recruited within the community. They were compensated at a rate of $3.50/hour. Individual subjects were required to have hearing threshold levels within ± 10 dB of audiometric zero (ANSI-S3.6.1969) at frequencies from 125 to 4.0 kHz. To obtain 52 subjects, over 100 were screened. Most rejections were due to hearing threshold levels in excess of 10 dB usually at frequencies above 3 kHz. A few subjects were "dropped" for failure to keep appointments and poor reliability in audiometric measurement. Four subjects were withdrawn during the actual noise exposure. Two were withdrawn because of parental concern and two because of bizarre audiograms.

Exposure to Noise

Subjects were exposed in groups of two in one room on the second floor of a two-story structure. In this room loudspeakers had been placed strategically so as to achieve a constant sound field in the space occupied to the subjects. Noise generating equipement and test facilities were located on the ground floor. A-weighted levels and octave-band levels were measured at several locations. Median values are reported elsewhere in this paper. Range of levels in the space occupied by the subjects did not exceed 2 dB. Thus, the levels reported here are accurate to ± 1 dB. Measured center frequencies were 63, 125 and 250 Hz (± 10 Hz), and the rejection rate was 24 dB/octave. . .

The noise exposure room was equipped with 2 cots, 1 desk, a refrigerator and toilet facilities. Subjects read, slept, played cards, etc.

Auditory Measurements

Pre-exposure measurements of auditory sensitivity were made daily over at least a 2-day period which preceeded the noise exposure. Earphone placement was also varied so as to achieve at least five pre-exposure measurements of auditory sensitivity over the frequency range of 0.25-4.0 kHz. The mean of these measures and a final pre-exposure audiogram constituted the pre-exposure measurements. Auditory sensitivity was measured by means of sweep-frequency (Bekesy) audiometry (Demlar, model 120). Below 250 Hz, auditory sensitivity was measured at 90, 125 and 180 Hz by means of fixed frequency Bekesy audiometry (Grason-Stadler, model E800). The tone was gated with an on time of 250 ms and a duty cycle of 50%. For the last 24 hours prior to the noise exposure, subjects were required to wear an ear plug (EAR) in the test ear so as to insure that the test ear was rested. The ear plug provided at least 20-40 dB attenuation.

During the noise exposure auditory threshold were measured during periods of quiet interspersed within an exposure. At a prescribed time, a subjects was removed from the noise for about 10 min. during which thresholds were measured. Thus, threshold shifts reported in this paper were recorded at post-exposure times between about 2 and 10 min. and hereafter are called 4 min. (TTS_4). The time spent away from the noise was always recorded and the total duration of the exposure was corrected.

RESULTS

Growth of TTS

Figure 1 (Panel B) shows TTS for the 90 dBA condition as a function of the duration of the exposure with the center frequency of the noise as the parameter. With the

exception of one irregularity (63 Hz condition at 4-7 hours) TTS increases throughout the exposure. The 3 to 4 dB differences between conditions are not particularly meaningful inasmuch as the range of TTS values is 8-10 dB between 4 and 8 hours of exposure. The 63 Hz condition deserves additional comment. The datum point at 8 hours is the result of a continuous exposure whereas the other data points reflect periods away from the noise when TTS was measured. In addition, the 63 Hz noise at 90 dBA interfered with the audiometric testing and therefore had to be attenuated for every test. Accordingly, the duration of the exposure for the 63 Hz condition is only an estimate.

Figure 1 (Panel A) shows TTS for the 84 dBA condition. These results shown on Figure 1 are not at all straightforward and are difficult to describe in simple terms. For the 250 Hz condition, TTS is 8 dB after the first 2 to 8 hours of the 24-hour exposure, and then increases to 14 dB between 12 and 24 hours. At the 125 Hz condition, TTS is inly barely measureable after 4 hours of exposure, and then appears to increase systematically from 4 to 12 hours., and then decreases. TTS for the 63 Hz condition increases between 2 hours and 12 hours, and then decreases.

It may be worthwhile to rank-order the three bands of noise with respect to TTS. For an exposure of 4 hours, the 250 Hz band produces the most TTS, then the 63 Hz band, and finally the 125 Hz band. At a duration of 12 hours, the 63 Hz band produces the most TTS followed by the 250 Hz band, and finally the 125 Hz band. In different words, for the 84 dBA exposure the rank-ordering changes as the duration changes. It is not clear whether the differences between the conditions of Figure 1 (B) are indicative of variability or "real" frequency effects.

Recovery of TTS

Figure 2 (Panel A,B,C) shows recovery from TTS. These recovery curves show at least one consistent result, namely that recovery after the 24-hour exposure at 84 dBA requires more time than recovery after the 8-hour exposure at 90 dBA. This result is particularly striking for the 250 Hz and 125 Hz exposures where the 24-hour exposure at 84 dBA produces significantly less TTS than the 8-hour exposure at 90 dBA. Thus, the duration of the exposure is clearly a determinant of the rate of recovery of TTS.

Data of Figure 2 show also that for all conditions recovery to within 2 dB of pre-exposure values occurred within 48 hours for the 24-hour exposure at 84 dBA and within 12 hours for the 8-hour exposure at 90 dBA.

Another possibly remarkable feature of the recovery data is shown on Panel B of Figure 2. Note that for the 125 Hz condition TTS actually increased by 4 dB between post-exposure times of 4, 8 and 12 hours. It is not clear whether this increase respresents measurement error or is an indication of a further deterioration of the ear.

Whereas a rank-ordering of the exposures in terms of the growth of TTS produced unreliable results expecially for the 84 dBA exposures, a rank-ordering in terms of recovery produces a straihtforward result. That is, at post-exposure times of 2-12 hours the exposures were equivalent. This equivalency asumes that differences between conditions of 2 dB are not meaningful. Similarly, for the 84 dBA exposure the differences between exposure conditions are within $\pm$ 2 dB between 1-12 hours of recovery. It is possible that at 24-hour and a 48-hour post-exposure, recovery is not complete for the 250 Hz exposure. At these long recovery times whee TTS is less than 5 dB, differences between conditions are not meaningful.

Spread of TTS Along the Frequency Dimension

Figure 3 shows the "TTS audiogram" produced by the 84 dBA exposure (Panel A) and the 90 dBA (Panel B). The parameter is the center-frequency of the octave-band noise. The data in Panel A for the 84 dBA exposure are averaged across measurements made after 8, 12 and 24 hours of exposure whereas the data in Panel B are for an 8 hour exposure only.

Figure 3 has several noteworthy points. One is that for both the 84 and 90 dBA exposures TTS is largest between about 350 and 750 Hz, and not at the test frequency ½ to 1 octave above the center frequency of the noise. One possible exception is the 125 Hz exposure at 84 dBA where TTS is 9 dB at 125 Hz and 250 Hz and 7.5 dBbetween 250 and 500 Hz. A difference of 1.5-2.0 is not meaningful. A second point is that TTS is not measurable (5 dB) at test frequencies above 2.0 kHz except for the 63 Hz exposure at 90 dBA. In this case, TTS is equal or nearly so between 180 Hz and 750 Hz and then decreases with an irregularity or discontinuity at a test frequency of 2.0 kHz. For the 90 dBA exposures (Panel B) differences between conditions are most obvious at test frequencies below about 250 Hz. These differences reflect the spectrum of the noise. At test frequencies of 750 Hz and 1.0 kHz, however, the values of TTS are nearly identical. In other words, the TTS between about 750 and 1000 Hz is independent of the spectrum of the noise. Possibly, the only ½ to 1 octave shift may be at 500 Hz for the 250 Hz exposure.

TTS and the Level of Noise

A crude estimate of the relation between TTS at the test frequency of maximum shift and noise level can be obtained by a comparison of TTS after 8 hours exposure at 84 and 90 dBA:

	OCT-Band cf		
	63	125	250 Hz
TTS, 8 hours, 84 dBA	13	9	9
TTS, 8 hours, 90 dBA	17	14	18
Estimated Slope	0.67	0.83	1.5

As shown, TTS increased when the noise level was increased from 84 to 90 dBA. The magnitude of the increase appears to be related to the center frequency of the noise. That is, the slope increases from 0.67 at 63 Hz, to 0.83 at 125 Hz, and to 1.5 at 250 Hz. A more detailed treatment of the relation between noise level and TTS is hampered by the absence of data for a 24 hour exposure at 90 dBA.

DISCUSSION

Growth of TTS

Previous TTS data from human subjects exposed to noise for 8-24 hours (see MiHs et al., 1979; Patterson et al., 1977) would suggest that TTS would increase for about 8-12 hours and then reach a plateau or asymptote. Discontinuities in the growth curves would

also be expected, particularly a 3 dB or so "overshoot" of the plateau or asymptote. Thus, the data reported on Figure 1 are consistent with the results of previous experiments and a priori expectation. Some of the results of the 84 dBA exposures are difficult to discuss, particularly the shapes of the growth curves during the first 4 hours of exposure and when one compares the data points at 4 hours for 125 Hz and 250 Hz. Moreover, it is difficult to assign the differences in the shapes of the growth curves for the 84 dBA exposures to procedural artifacts. Perhaps a sampling error is operating and a much larger N is needed.

Recovery

The recovery data were consistent in many respects with previous data and with a priori expectations. The time required for recovery to 0-TTS was influenced significantly by the duration of the exposure. Recovery from the 84 dBA exposure for 24 hours required 24-48 hours whereas recovery from the 90 dBA exposure for 8 hours was usually complete within 12-24 hours. This result is consistent with results of others (Mills et al., 1970; Ward, 1970; Nixon et al., 1977). Thus, an earlier hypothesis (see Ward, 1963; Kryter et al., 1966) that the recovery from TTS is independent of how the TTS is produced is becoming increasing difficult to accept.

An issue in human TTS experiments centers around the risks presented to the subjects. In other words, can human subjects be used without creating a significant risk of permanent hearing loss and permanent injury to the inner ear? The present results strongly suggest that the risk to the subjects is minimal. . For example, all subjects recovered to within 2 dB of pre-exposure thresholds within 12-48 hours after termination of the exposure. In other words, 0-PTS was produced. Not only was there an absence of PTS but rates of recovery were faster than those observed in animals who had 0-PTS but small losses of outer hair cells (see Eldredge et al., 1973). If the subjects of the present experiments had taken 3-7 days to recover, the possiblity of permanent injury to the inner ear could not be discounted.

Spread of TTS Along the Frequency Dimension

In most experiments where the exposure has a spectrally dominant peak, TTS is maximal ½ to 1 octave band above that peak. Sometimes a second maximum is observed at frequencies nearly 3 octaves above the peak (see Mills et al., 1979). In the present data the most striking features of the TTS audiograms were: 1) that the greatest TTS was observed between about 300 Hz and 750 Hz regardless of the center frequency of the exposure, and 2) with one exception TTS was not measured at test frequencies above about 2 kHz. The absence of the dominant peak in the TTS audiogram may be due to a number of factors. One of the more likely possibilities for maximal TTS in the 300-750 Hz region is the difference in auditory sensitivity between, for example, 90 Hz and 750 Hz. This difference may be as large as 50-55 dB and is indicative of the acoustic properties of the external and middle ear (Zwislocki, 1979). Thus, while the exposures at 63, 125 and 250 Hz had spectral peaks when measured in the sound field, the spectral peaks when measured at the input to the cochlea would be much less prominent.

While the absence of TTS at frequencies above 2.0 kHz for the 125 and 250 Hz exposures is inconsistent with expectations based on previous data (Burdick et al., 1978; Patterson et al., 1977; Mills et al., 1979) the notch in the TTS audiogram at 2.0 kHz for the 63 Hz exposure corresponds exactly to a priori expectations. Again, there are no obvious explanations for this mixed support of a priori expectations.

On the one hand, the impression is that 90 dBA exposures at 63, 125 or 250 Hz are equivalent or nearly so when comparisons are made at the test frequency of maximum shift. Similarly, an 84 dBA exposure at 250 Hz and 83 dBA at 4.0 kHz are nearly equivalent to each other and to 90 dBA exposures at 63, 125 or 250 Hz. On the other hand, if exposures are judged not on the basis of the TTS (or PTS) at the frequency of maximal shift but on "TTS audiogram" then an entirely different ranking (most to least TTS) occurs as follows: 63, 125, 250, 2.0 kHz and 4.0 kHz. Stated differently, a much greater region of the cochlea is affected by 63 Hz than by 4.0 kHz, and then, the number of sensory cells and other elements at "potential risk" is significantly greater with low-frequency exposures.

TTS and Level of the Noise

Previous results from humans and animals (Burdick et al., 1977; Patterson et al., 1977; Mills et al., 1979) suggested after 8-24 hours of exposure TTS would increase about 1.5-2.0 dB/dB increase in noise level. Only the results for the 250 Hz exposure were consistent with this expectation. For the 63 Hz and 125 Hz exposures, the increase was only 0.67 and 0.83 dB/dB. It is not clear why TTS produced by 63 Hz exposures should increase at a slower rate than TTS produced by exposures at 250 Hz or higher. Additional data are required.

Extrapolations from the data suggest that to produce TTS's of less than 5 dB at any test frequency after 8-24 hours of exposure, octave-band levels of noise should be about 104 dB SPL (78-80 dBA) at 63 Hz, 98 dBA SPL (78-80 dBA) at 125 Hz, and about 87 dB SPL (78-80 dBA) at 250 Hz. Additional data are needed to obtain precise estimates of these "safe" or "critical levels". "Safe" or "Crictical levels" have been defined previously (Mills et al., 1979) for octave-bands spaced across the frequency range of 0.5 to 4.0 kHz. Thus, estimates of critical levels now cover the frequency range from 63 Hz to 4.0 kHz.

Measurement of Sound Levels

The present results, particularly for the exposures at 90 dBA, support the use of the A-weighting network. That is, for octave-band noises centered at 63, 125 or 250 Hz and equated in terms of A-wighted SPL, the growth and decay of TTS were not meaningfully different. Moreover, comparisions to TTS data produced by an exposure to an octave-band centered at 500 or 1000 Hz support the validity of the A-weighting network for center frequencies from 63 to 1000 Hz. For octave-band noises centered at 2.0 or 4.0 kHz, however, the A-weighting network appears to be inaccurate. Octave-band noises centered at 2 kHz or 4 kHz at a level of 83-85 dBA produced growth curves for TTS that were similar to those observed for octave-band noises of 90 dBA centered 500 Hz and below. Thus, as stated previously (Mills et al ., 1979) the A-weighting network does not give sufficient emphasis at 2.0 and 4.0 kHz by about 5 dB or so. The argument that the A-weighted network places insufficient emphasis at the higher frequencies assumes that a hearing loss of, for example, 20 dBA at 4.0 kHz is as socially and medicall significant as a hearing loss of 20 dB at 0.5 or 1.0 kHz. Currently, this assumption is not accepted by most of the methods used to assess hearing handicap.

Miscellaneous Comments on Auditory Effects

An unsolicited response from many of the subjects involved the sensation of a "tremondous pressure build-up" in the ear, particularly for the 63 Hz exposure. These informal reports confirmed the impressions of the experimenters. Interestingly, while the non-acoustic sensation was similar to the "pressure build-up" in the middle ear associated with, for example, altitude changes, the sensation could not be eliminated by opening of

the eustachian tube by swallowing, yawning, or by the Valsalva technique. A more vigorous evaluation of the "sensation" is planned in future experiments.

An additional point of some interest involves a number of basic questions concerning the relation between anatomical integrity of the cochlea and the detection of auditory signals. For example, we can not specify that the "unaffected" basal region of the cochlea was not used to detect tones less than 500 Hz. A complex masking paradigm is needed to eliminate the possibility of "basal region detection". Also, PTS's can be as small as 10 dB at 250 Hz or 125 Hz and as many as 50-65% of the outer hair can be missing (Bredburg, 1968). Thus, it is possible that our estimates of the effects of the low-frequency noises are gross underestimates, and that certain physiological or other criterion measures would be better.

Although not required by the contract, one of our staff (R.A. Schmiedt) experimented with the effects of a 63 Hz noise as indicated by single-unit and gross physiology of N VIII, and sensory cell counts. In regard to the latter, gerbils had large injuries (OCH's) in the apical region and in the base. Single-units ranged from normal in most respects to abnormal tips of tuning curves and bizarre patterns of two-tone suppression. These results suggest additional physiological study of the effects of a 63 Hz noise, and that a 63 Hz noise may produce effects unlike those associated with higher frequency noises. In other words, we are reluctant at this stage to claim that the effects of a 63 Hz noise are well documented or predictable given the data currently available.

PART II. NON-AUDITORY EFFECTS

INTRODUCTION

Noise is one of many stressors which can induce increases in certain cortical, autonomic and endocrine activities in animals known as the general adaptation syndrome (Selye, 1976). Such increases are mediated primarily by the sympathetic nervous system through catecholamine secretion and by hypothalamo-pituitary stimulation of adrenocortical hormone release. Animal studies suggest an acute elevation in the plasma level of these substances during loud noise, and the development of hypertension under chronic exposure (Moller, 1978; Peterson et al, 1981). Generalizations from the animal to the human situation are not straightforward. Whereas animals react to loud noise with fear, the cognitively assigned meaning ascribed to the same stimulus can significantly modify the response in humans (Follenius et al., 1980; Levi, 1967; Frankenheuser and Johansson, 1976). In addition, much of the human data is restricted to noise exposure where the potential for acoustic injury to the inner ear is minimal. While it has been suggested that a noisy environment contributes to the development of coronary vascular disease, hypertension, and gastrointestinal disorders (Selye, 1976; Metz et al., 1978; Ortiz et al., 1974; Jonsson and Hansson, 1977), the data on non-auditory effects of noise in humans is contradictory and no general agreement exists (Moller, 1978; Peterson et al., 1981; Follenius et al., 1980). As part of a larger study of low frequency noise, the heart rate, blood pressure, urinary catecholamine excretion and plasma cortisol of healthy subjects were examined in relation to the intensity and duration of short-term exposure.

MATERIALS

Thirty-five male college students (age 18-22) were screened from a population of over 100 as follows: (1) hearing threshold levels within 10 dB of ANSI-S3.6 at frequencies from 125 Hz to 4.0 kHz, (2) good health, with no current diseases or drug ingestion, (3) reliability of audiometric measurements, and (4) freedom from observable vasovagal reactions to venipuncture.

METHODS

A group of 18 were exposed to 84 dBA of octave band noise centered at 63, 125 or 250 Hz for 24 hours. Due to prolonged temporary threshold shifts (TTS), exposure of a second group of 17 at 90 dBA was limited to 8 hours. Four subjects taken from this 90 dBA group were confined for 24 hours without noise, and served as controls.

The subject's oral intake was regulated for 12 hours prior to confinement to eliminate stimulants (e.g. nicotine, caffeine, alcohol) from the urine and blood. Thirty-two ounces of water were ingested between the morning check-in and preexposure samples to insure adequate urine flow, and a high fluid intake was continued during the testing. A standardized meal was served in the 4th and 10th hours of noise exposure. Two subjects were observed per session, in a small furnished apartment with amplification arranged to produce a constant sound field ($\pm$ 1 dB). Restricted to sedate pursuits, the subjects usually slept or read.

The effects of noise on the sympathetic nervous system were inferred from measurements of heart rate and blood pressure, and from the urinary excretion of epinephrine(E) and norepinephrine(NE). Heart rate (HR) and blood pressure were recorded prior to the onset of noise, and thereafter as indicated in Fig. 1. Measurements were taken from an automated blood pressure cuff and sound transducer equipped with a digital readout (Somnatronix, model 300). Subjects voided one hour prior to exposure, and then urine samples were collected at 0,1,and 2 hours. The urinary output of each subject in the remaining 6 hours (90 dBA) or 22 hours (84 dBA) was pooled and reported as a mean excretory rate. These were assayed for E and NE by a modification of the fluorometric technique of Crout, with a coefficient of variation of 7.2% for NE and 22% for E.

Plasma cortisol was measured from venipuncture samples which were obtained just prior to noise exposure (9 a.m.) and then 1 and 24 hours later. The radioimmunoassay (Micromedics Systems) had a coefficient of variation of 7%.

RESULTS

The auditory effects of low frequency noise are relevant to this paper in that the 90 dBA subjects TTS's required up to 48 hours for recovery. The mean TTS in the 90 dBA group at 8 hours was 15 dB, exceeding the mean increase in the 84 dBA subjects at 24 hours by 5 dB. The auditory effects differed in certain aspects between 63, 125 and 250 Hz octave-band noise, but no significant dissimilarites were observed in the physiologic parameters. The data presented herein represents an averaging across the 3 frequencies.

The mean HR and mean arterial pressure (MAP, diasystolic pressure plus 1/3 pulse pressure) (Abel and McCutcheon, 1979), of the 84 and 90 dBA groups are shown in Figure 1. Both the systolic and diasystolic parameters increased in 12/17 of the 90 dBA subjects, resulting in a rise of 5 mm of Hg in the group's MAP between the 0 and 8 hour measurements. Their mean HR fell 7 beats per minute (BPM) between the 0 and first hour measurements, with 14/17 of the subjects demonstrating this pulse slowing. The mean HR consistently rose thereafter but did not reach the initial level of 71 BPM. In contrast, the 84 dBA group's MAP decreased 5 mm of Hg during the first hour, and thereafter varied from 88 to 94 mm of Hg. Their 24 hour MAP was 4 mm of Hg less than pre-exposure. The mean HR of this group ranged from a high of 68 (meal) to a low of 58 (sleep), with no clear trend. The control group (data not shown) demonstrated little change during their quiet confinement, with both HR ($\pm$ 5 BPM) and MAP ($\pm$ 2 mm of Hg) essentially unchanged between the 0 and 24 hours measurements.

The urinary excretion of E and NE for the 1st, 2nd and mean of the remaining 6 hours (90 dBA) or 22 hours (84 dBA) in noise are shown in Tables 1 and 2, as normalized to the excretory rate of the one hour prior to noise exposure. A decrease in E was noted in approximately two-thirds' of subjects in both noise groups between the 0 and 1 hour measurements, with a subsequent rebound to near initial levels in the 2nd hour. The lower mean E excretion for the remaining 22 hours of exposure in the 84 dBA subjects may reflect the depressed basal secretion rate of their usual 9 hours of sleep. The 90 dBA subjects' final excretion rate does not contain any sleep-produced bias, and demonstrated little change.

Norepinephrine excretion in the 84 dBA group increased slightly over the first 2 hours to 16% above pre-exposure, but the mean of the remaining 22 hours differed from

the initial level by only 2%. In contrast, this parameter decreased slightly in the 90 dBA subjects during the first 2 hours to 22% below pre-exposure, and then stabilized near this level for the remaining 6 hours. The reason(s) for the contrasting 0 to 2 hour patterns between the groups is not known.

None of the trends noted in E and NE were statistically significant. There was wide variability in the excretion of catecholamines between the subjects, and the standard deviations for the normalized values presented in Table 1 and 2 averaged 50%. This may be partially attributed to differences in their weights (medium = 73 Kgs, range of 62 - 100 Kgs). In addition, the subjective response of each individual to the noise and confinement was not quantified, but has been reported to significantly influence catecholamine response (Frankenheuser and Johansson, 1976; Metz et al., 1978).

Plasma cortisol (C) was measured at 9 a.m. (just prior to noise exposure), and then at 1 and 24 hours of confinement (Fig. 2). A subgroup (N=9) of the 90 dBA subjects were restricted to the cots for a "quiet hour" prior to the pre-exposure sample and the onset of noise. Their mean C decreased 6.75 ug/dl, which might be interpreted as some combined effect of the normal circadian morning decline and the non-stimulating environment. During the first hour of noise exposure, the downward C trend was reversed, with 3 ug/dl mean increase in both the 90 dBA and 84 dBA subjects. These changes are significant in view of the expected morning decline in C demonstrated by the control subjects. Twenty-four hours after the onset of noise, C was elevated in both the 84 and 90 dBA groups. The 90 dBA subjects had been out of the noise for 16 hours, but still exhibited a significant increase (P. 05) from the previous days' 9 a.m. sample.

DISCUSSION

A deceleration in HR and rise in BP with the onset of loud noise has been observed in animals and, less consistently, humans (Moller, 1978; Hawel and Starlinger, 1967). In this study, these trends were observed only in the 90 dBA group and were not statistically significant.

It has been stated that increased E release is the strongest reaction to noise in humans, with NE being less responsive (Ortiz et al., 1974; Hawel and Starlinger, 1967). However, the urinary E excretion decreased in a majority of subjects in both the noise groups during the first hour of exposure, and never showed a significant elevation during the entire period of noise and confinement. These results support Follenius et al, who demonstrated no significant change in plasma or urinary catecholamines to alternating 99 and 45 dBA broad band pink noise exposure, but observed a wide variability within the subject group (Follenius et al., 1980).

In both the 90 and 84 dBA subjects, the normal morning C decline was reversed within one hour of noise exposure. It remained significantly elevated in the 90 dBA group 24 hours later. These observations are consistent with previous reports that plasma C rises acutely in both humans and animals in response to aversive noise levels (Moller, 1978; Follenius et al., 1980). Cantrell has observed this C elevation to persist during chronic exposure in humans (Cantrell, 1974).

Differences in the magnitude of the physiologic response to noise between animal and human subjects has been ascribed to the absence of a perceived threat from this

"stressor" by the humans Metz et al., 1978). For instance, it has been reported that noise, light and assigned tasks have less influence on human catecholamine excretion than does the subject's attitude towards the experimental paradigm (Levi, 1967). In our study, the 84 dBA group did not report the low frequency noise to be aversive, and their uninterrupted reading, sleep and other activities tend to support this. The 90 dBA subjects were initially bothered by the noise, though most claimed to become tolerant within a few hours and some even fell asleep. Their significant C elevation 16 hours after noise termination indicates that the hypothalmo-pituitary system may not adapt as readily.

The non-auditory effects of low frequency noise exposure seem consistent with those recently reported at higher frequencies (Follenius et al., 1980; Cantrell, 1974). The changes observed in this study are conservative, as the subjects were subjected only to the stresses of constant noise and 3 venipunctures. The potential for intermittent/unpredictable noise, task demands and other distractions to increase these somatic responses has been well documented (Selye, 1978; Levi,1967; Frakenheuser ,and Johansson, 1976; Cantrell, 1974). Whether the C elevation in both the noise groups, and the BP trend of the 90 dBA subjects, would have persisted or increased with repeated exposure is not known.

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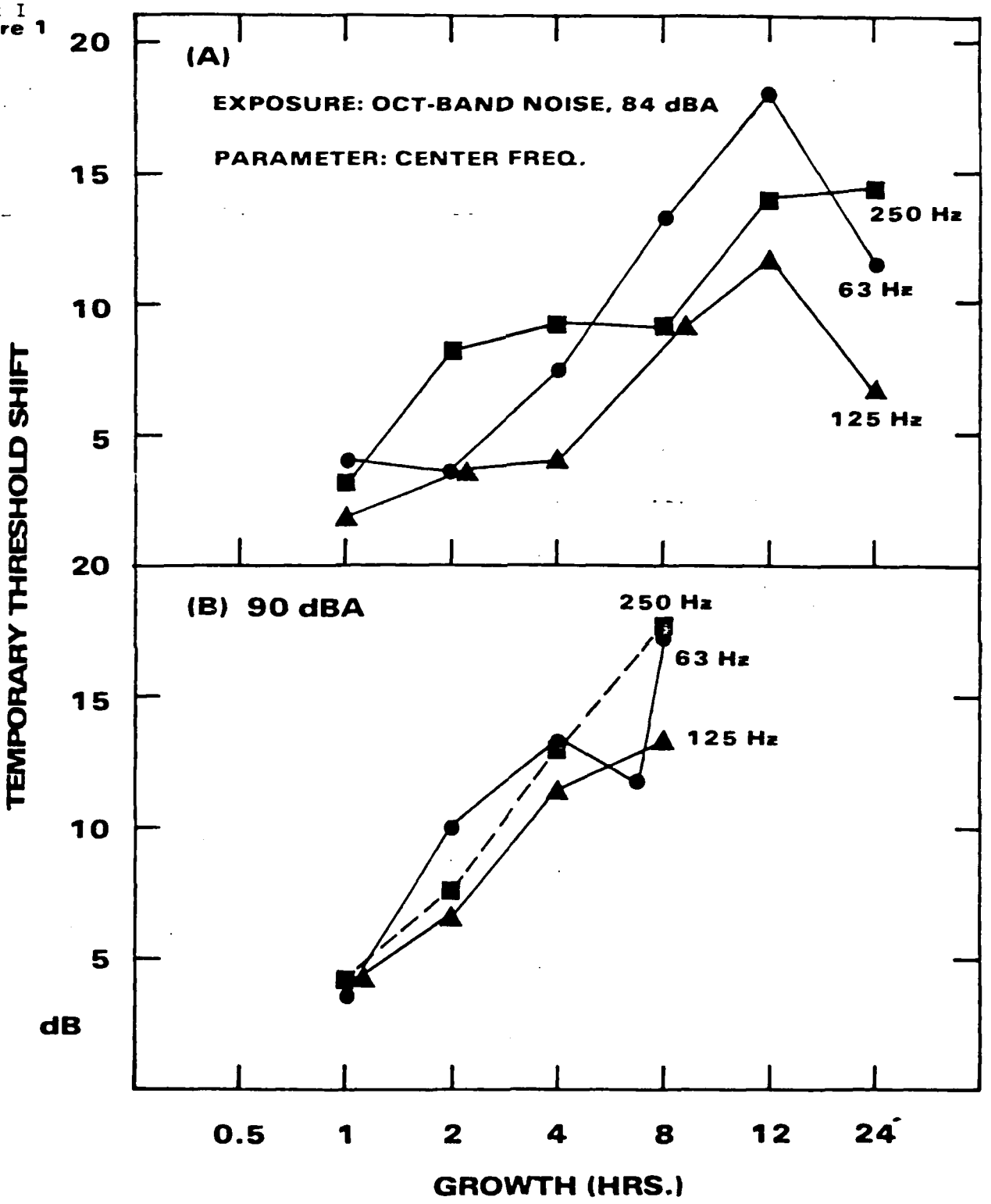
LEGENDS
(Part I.)

Figure 1. TTS as a function of the duration of exposure. The A-weighted sound pressure level of the noise is 84 dB in Panel A (top half) and 90 dB in Panel B (bottom half). The parameter is the center frequency of the octave-band noise. The noise had a rejection rate of 24 dB/oct. N=8 males datum point except at 63 Hz, 8 hours where N=6.

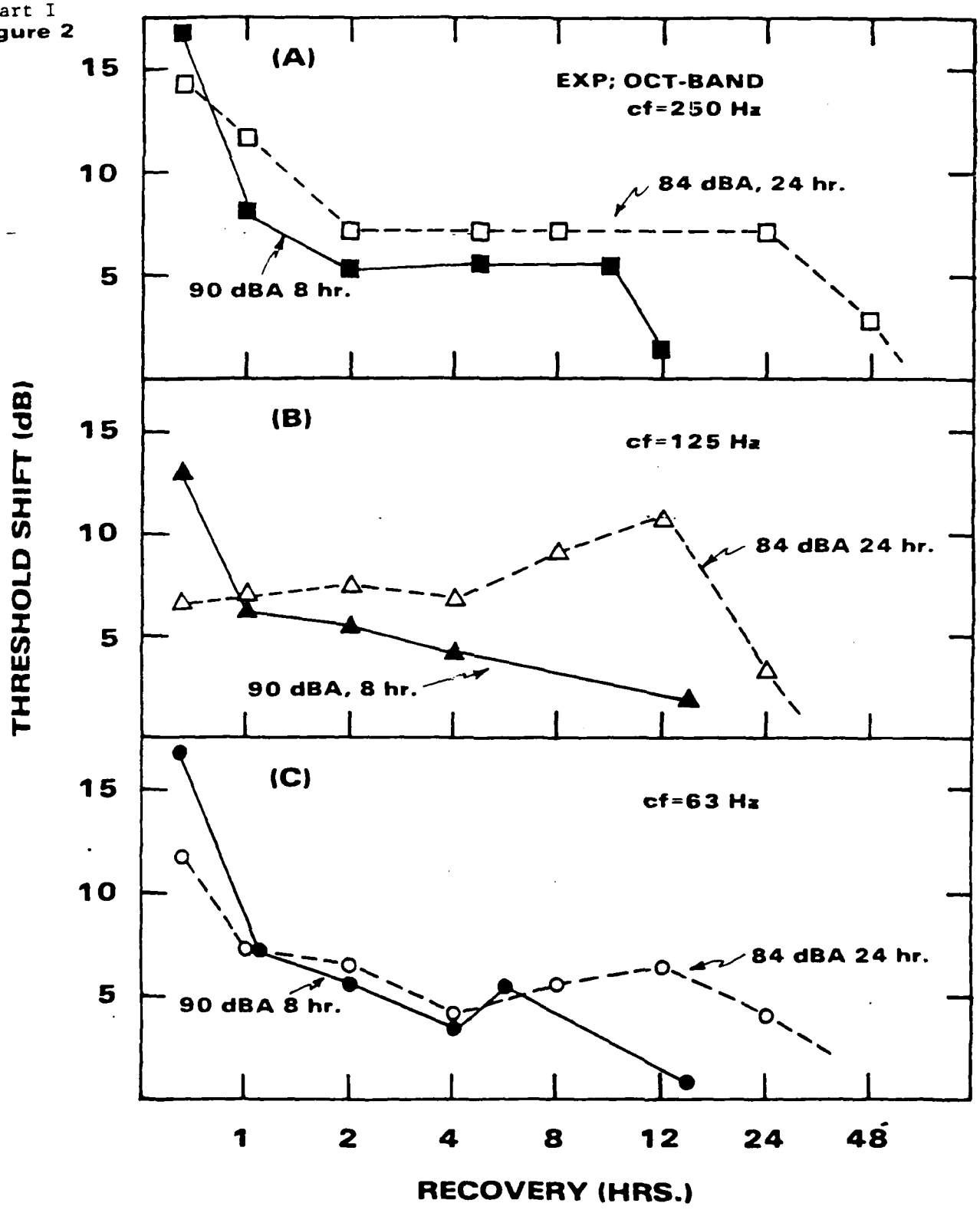
Figure 2. TTS at the test frequency of maximal shift after exposure to an octave-band noise for 8 hours or 24 hours. Panel A, 250 Hz; Panel B, 125 Hz; Panel C, 63 Hz.

Figure 3. TTS at different test frequencies. Panel A shows results for the 84 dBA exposure. Results have been averaged across 8-, 12- and 24- hours of exposure. Panel B shows results for 90 dBA exposure for 8 hours. Parameter in both Panel A and Panel B is the center frequency of the octave-band exposure.

Part I
Figure 1

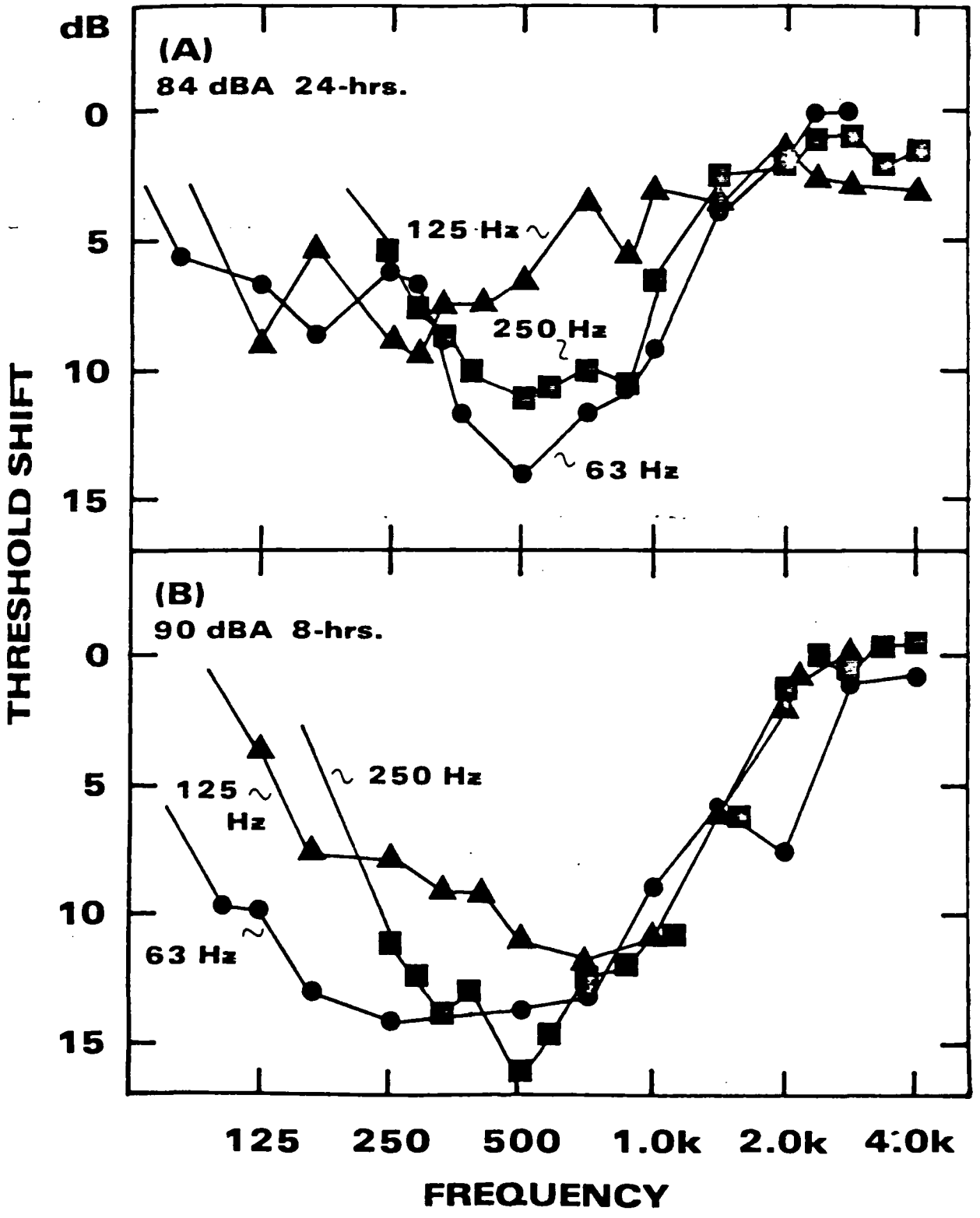


Part I
Figure 2



Part I

Figure 3



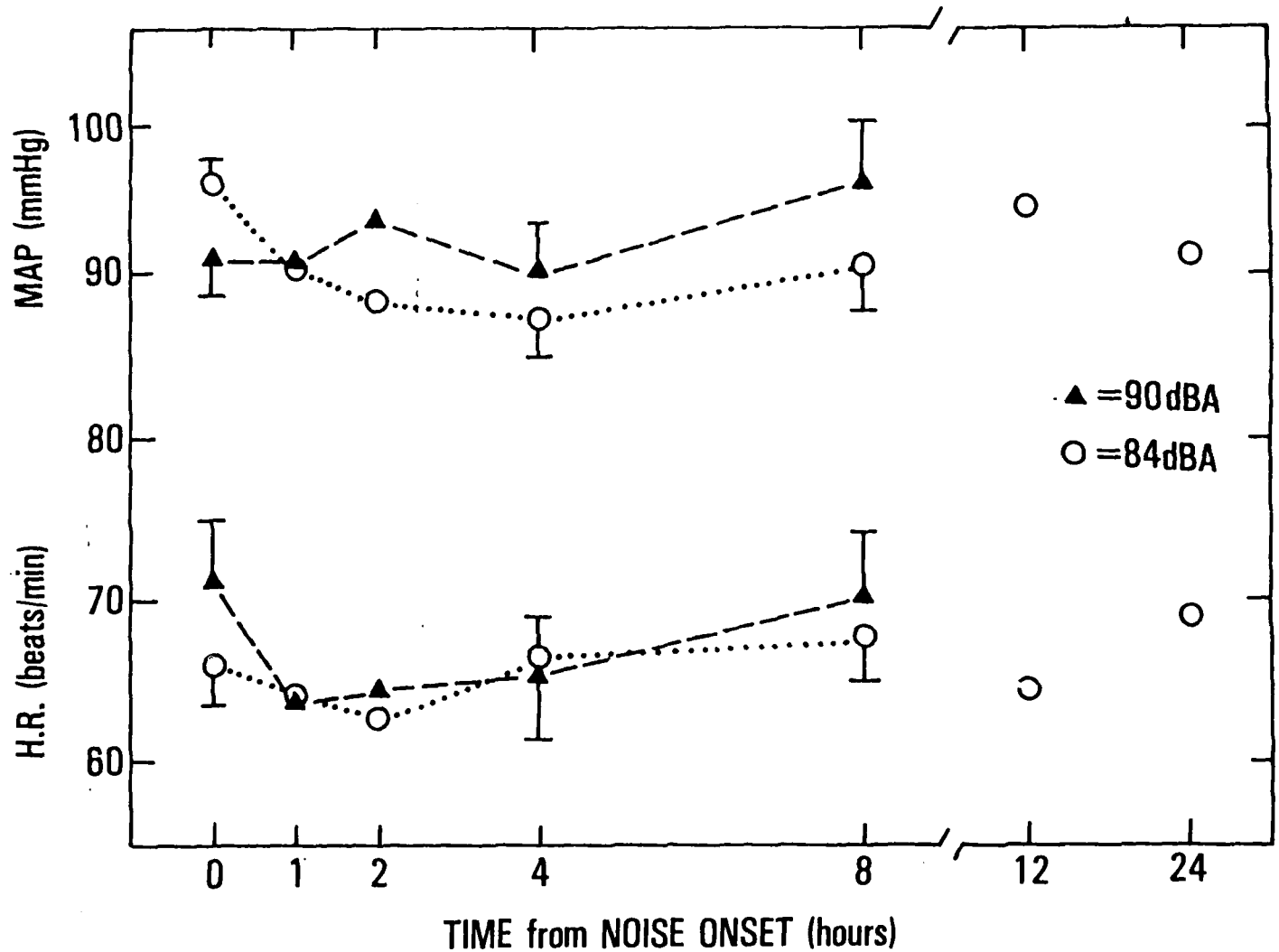
LEGENDS
(Part II.)

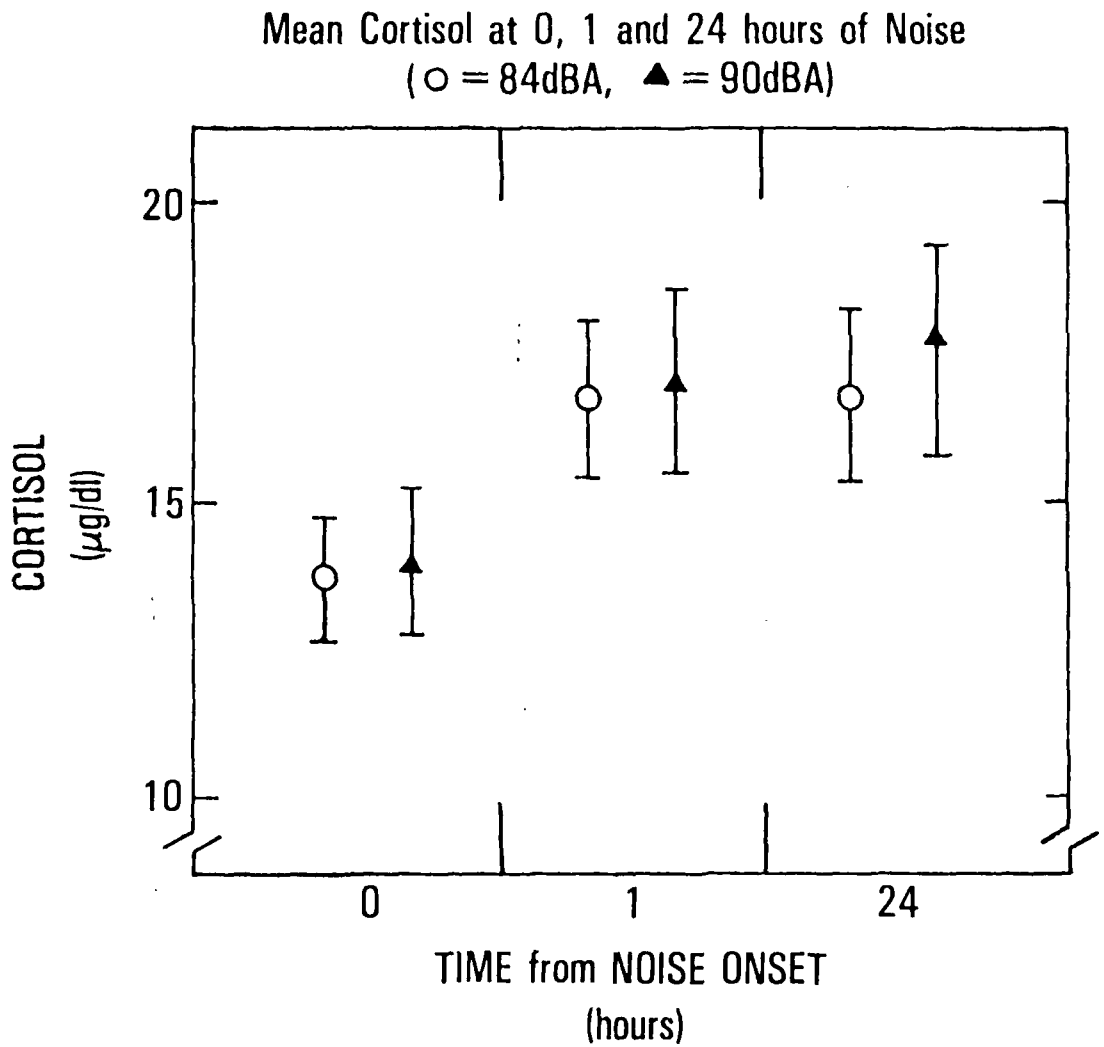
Figure 1. Mean arterial pressures (MAP) and mean heart rates (HR) of the 90 dBA and 84 dBA groups from pre-exposure through 8 or 24 hours in the noise. Standard errors indicated at 0 and 8 hours.

Figure 2. Mean plasma cortisols (with standard errors) just prior to noise exposure (9 a.m.), and at 1 and 24 hours later (90 dBA = , 84 dBA = O).

Table 1. Mean urinary E excretion for the one hour ("0") prior to noise exposure, with the first ("1"), second ("2") and the remaining hours in noise (6 hours for 90 dBA and 22 hours for 84 dBA) as normalized to the pre-exposure hour ("0").

Table 2. Mean urinary NE excretion for the one hour ("0") prior to noise exposure, with the first ("1"), second ("2") and the remaining hours in noise (6 hours for 90 dBA and 22 hours for 84 dBA) as normalized to the pre-exposure hour ("0").


MAP (mmHg)
100
90
80
H.R. (beats/min)
70
60
▲ =90dBA
O =84dBA
0
1
2
4
8
12
24
TIME from NOISE ONSET (hours)


Mean Cortisol at 0, 1 and 24 hours of Noise
(○ = 84dBA, ▲ = 90dBA)
CORTISOL
(μg/dl)
20
15
10
0
1
24
TIME from NOISE ONSET
(hours)

MEAN HOURLY EPINEPHRINE EXCRETION IN URINE
(data normalized to "0" hour)

Part II
Table 1

Time in Noise (hours)

Noise Level	0	1	2	Remainder
84 dBA	1.00	.85	.97	.62
90 dBA	1.00	.69	.98	1.06

MEAN HOURLY NOREPINEPHRINE EXCRETION IN URINE
(data normalized to "0" hour)

Part II
Table 2

Time in Noise (hours)

Noise Levels	0	1	2	Remainder
84 dBA	1.00	1.05	1.16	.98
90 dBA	1.00	.82	.78	.82